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Comment:

In the last 10-15 years there has been a steady increase in the number cases of autism spectrum disorder (ASD) in the United States. Recent research has shown that dysfunction of the human mirror neuron (MN) system may contribute to causing ASD. The MN system refers to neurons that fire both when an action is performed and when it is observed. This link between action and perception develops over the first few years of a human's life. This paper suggests that the development of the MN system may be disrupted by exposure to electromagnetic radiation (EMR) during infancy and early childhood.

As the number of ASD cases has increased over the last decade, so too has the use of cable, satellite, and mobile phones in the US. Exposure to EMR waveforms can come from these sources, as well as common household appliances such as microwaves, refrigerators, and computers. Research has shown that the central nervous system reacts to exposure to EMR by resonating to the electrical or magnetic fields in the EMR waves. This resonance can cause motor tremor and changes in reaction time and sleep patterns. While these effects may be barely detectable in fully developed adults, exposure during children's development could have much more serious effects on their neural functions.

The article cites two specific studies to support its claim that environmental EMR can effect the central nervous system. In one study, subjects were exposed to low-frequency magnetic fields, at strengths associated with common household appliances. The study concluded that the magnetic fields effected postural tremor, or the ability to maintain the position of the limb, by increasing low frequencies in the tremor. A second study found that when exposed to EMR frequencies associated with cell phones while performing a cognitive task, individuals showed a decrease in task-related memory function.

The correlation between EMR exposure and MN dysfunction should be researched further by comparing different geographical areas or home environments with varying amounts of EMR exposure. The mechanisms through which EMR exposure during early development affects the MN system also need to be investigated further to provide evidence for EMR's role in causing ASD.



Out of time: A possible link between mirror neurons, autism and electromagnetic radiation

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Summary Recent evidence suggests a link between autism and the human mirror neuron system. In this paper, I argue that temporal disruption from the environment may play an important role in the observed mirror neuron dysfunction, leading in turn to the pattern of deficits associated with autism. I suggest that the developing nervous system of an infant may be particularly prone to temporal noise that can interfere with the initial calibration of brain networks such as the mirror neuron system. The most likely source of temporal noise in the environment is artificially generated electromagnetic radiation. To date, there has been little evidence that electromagnetic radiation poses a direct biological hazard. It is clear, however, that time-varying electromagnetic waves have the potential to temporally modulate the nervous system, particularly when populations of neurons are required to act together. This modulation may be completely harmless for the fully developed nervous system of an adult. For an infant, this same temporal disruption might act to severely delay or disrupt vital calibration processes.

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Recent evidence suggests that a dysfunction of the human mirror neuron (MN) system could underlie the complex pattern of deficits associated with autism spectrum disorder (ASD) [1–5]. Mirror neurons take their name from motor cortex cells, first discovered in monkeys [6], that fire both when an action is performed and when it is observed. In humans, the MN system is a late-evolved brain network – including frontal, parietal and temporal areas – that closely links the perception and production of action [7,8]. Achieving a tight coupling between motor areas and perceptual/cognitive

areas is thought to be a precursor for the development of many uniquely “human” mental functions, such as theory of mind [9], language [10] and high-level perception [11,12]. The overlap between such functions and the pattern of deficits found with ASD, particularly social interaction and communication problems, first prompted the idea of a possible link [1]. As evidence mounts for the role of MN in explaining ASD two issues remain. First, what exactly is going wrong with the MN system? What is the nature of the MN dysfunction? Second, what could be leading to such a rapid rise in the occurrence of the dysfunction, given that the dramatic increase in ASD needs to be explained.

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In this paper I will argue that *temporal disruption* early in development could be to blame for the MN dysfunction. Previously, it has been suggested that a very general advantage to having a MN system might be to make representations and processes for manipulating and controlling time-varying information available outside of the motor system [13,14]. It is uncontroversial that time is a crucial component of any stored motor pattern. For models of perception and cognition, however, the temporal dimension is typically either ignored or poorly understood [15]. The suggestion is that the MN system may have used motor programmes to help later-evolved areas of the brain come to terms with time [14].

Thus, both the basic architecture of the MN system – the need to coordinate motor and perceptual areas – and the information content, may involve time-critical processes. Crucially, this suggests that the MN system must be highly adaptive. That is, while the basic architecture of the system and its interconnections may be in place at birth, it is only during early development, as the infant begins to act-on and experience the environment, that the precise temporal coupling between action and perception can be established. Clearly, during the first few years of life, as the component motor and perceptual systems rapidly develop, the MN system must be in a continual state of calibration and change. My argument is that during this process of calibration the MN system will be particularly susceptible to temporal disruption of any of its underlying neural circuits.

To explain how temporal disruption might be involved, I want to use a parallel example from linguistic development. It has been shown that at birth the auditory system of the human is capable of detecting differences among the speech sounds used in any of the world's languages [16,17]. Shortly after birth, as an infant is exposed to its native language, there is rapid tuning of auditory responses such that between 6 and 12 months there is a preference only for those sounds that are used to make phonemic distinctions in that language [18–20]. Imagine how this process might be affected if, in addition to speech sounds, the infant was routinely exposed to systematic, but linguistically meaningless, auditory noise. That is, imagine filling your home with speakers and continually playing synthetic, meaningless pseudo-speech sounds. Could the phonemic system still calibrate itself? Could it achieve such calibration within the necessary time window, so as not to affect more general language processing? Note that I am not suggesting the optimal auditory environment for language development is silence. My question here

refers to sounds similar enough to real speech to interfere with calibration.

Returning to temporal disruption, the analogous situation would be one in which, during early development, the environment of the child was filled, not with auditory noise, but with temporal noise. That is, the developing nervous system of the child was exposed to systematic, but functionally meaningless, temporal modulations at the molecular, cellular or systems level. Again, we might ask whether calibration of the MN system would be possible under such circumstances or whether it could be achieved within some desired timeframe? The difference to the auditory case outlined above is that there is reason to believe that we are in the process of doing just that. More specifically, I want to suggest the presence of electromagnetic radiation, particularly in the home environment, while not posing a more obvious biological hazard, may be providing a significant source of temporal interference.

Electromagnetic radiation refers to the propagation of non-ionizing energy through space in the form of *time varying* electrical and magnetic fields. In the case of radio, television and telecommunications signals (e.g., mobile phones, cordless phones, and baby monitors), the propagation of EMR waves is intentional. In the use of many household electrical appliances, such as personal computers, microwave ovens and refrigerators, it is not. Important for the current argument is the idea that these temporally modulated waveforms might interact in some way with the human nervous system. Before returning to this point, I want to note that during the last 10–15 years the rise of cable, satellite and mobile telecommunications has greatly accelerated both the variety and the quantity of EMR sources in the environment. In Fig. 1 I have correlated the rise in autism in the US school system with the rise in sales of personal mobile phones during this same period. I am not suggesting that correlation equals causation, nor do I want to point the finger at a single EMR source – it seems more likely that poorly understood interactions in the EMR landscape could be to blame – but the time-scales of the growth in home electronics and communications and the rise of ASD at least raise the possibility that an environmental factor may be closer to home than we suspect.

I am well aware that despite a great deal of research, there is little compelling evidence that exposure to environmental EMR poses a direct biological hazard [21]. Nevertheless, it has been shown that the nervous system is responsive to the broad range of frequencies produced by electrical devices. This is true for the very low range associated

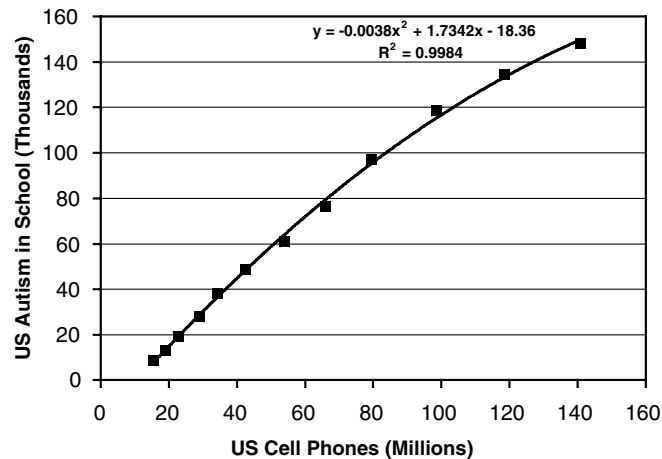


Figure 1 Correlation between children with a diagnosis of autism attending US schools and US cellular phone sales, during the period 1992–2003 [34,35].

with domestic appliances and power lines (10–100 Hz), to medium range radio frequency and microwave radiation used in FM radio, television and mobile communications (1–900 MHz) and the very high frequencies of microwave ovens and satellite communication (2–10 GHz) [22–27]. Such responsiveness is thought to take the form of a resonance within the nervous system to the electrical or magnetic fields in the EMR waveforms. Interestingly, sensitivity to these waveforms has been shown to be greater in networks than in single cells [22]. The impact of such resonance has been measured in terms of direct physical side effects, such as motor tremor [23], changes in reaction time [24] and changes in sleep pattern [25], as well as indirect modulation of EEG signals [24,26,27]. For an adult, fairly stable nervous system – the system that has typically been modelled or considered in the EMR hazard research [21] – such responsiveness may amount to little more than transitory, barely detectable perturbations, even in those individuals with particular “electrosensitivity”. For an infant nervous system, one that is struggling to temporally calibrate itself, these very same mild perturbations could be catastrophic.

Two recent examples will serve to illustrate how EMR has been shown to modulate the nervous system. In one laboratory study, individuals were briefly exposed to extremely low-frequency (50 Hz) sinusoidal magnetic fields at strengths typically encountered with common home appliances [23]. During exposure, postural tremor – the ability to maintain the position of a limb – was measured. It was found that a magnetic field “corresponding to those present in our modern daily environment” had a clearly detectable effect on postural tremor. Specifically, the magnetic field increased the pro-

portion of low frequencies in the tremor, an effect that could still be measured several minutes after exposure was terminated. Another study used EMR frequencies in the microwave range associated with mobile phones [24]. Post-exposure, behavioural measures and EEG signals were recorded during performance of a demanding cognitive task. Exposure increased the spectral power of a specific EEG frequency band (8–10 Hz) and led to a decrease in task-related memory function.

Bearing these examples in mind, let me state my argument more explicitly: I am suggesting that an underestimated side effect of EMR waveforms, the fact that they can modulate the nervous system in time, may block or retard the normal development of the human MN system. In turn, such a dysfunctioning MN system may give rise to the complex pattern of deficits associated with ASD. Why would only some individuals be affected by EMR? The rise in ASD, while growing exponentially, is still below 1% of the population. I can also offer no explanation for the 4:1 male prevalence bias. Clearly other factors must influence susceptibility. Such factors might include specific combinations of EMR signals in the environment, individual or sex differences in normal MN systems development (i.e., slower development leads to more exposure) or previously implicated medical or genetic risk factors, such as epilepsy or fragile x syndrome.

My focus on the MN system has been prompted by a number of factors, notably my own more general research interests in perception and action [13], the common involvement of time across these domains, and recent theoretical and empirical links between the MN system and ASD [1–5]. It remains wholly possible that temporal disruption as I have described it is not limited to the MN system.

Indeed, Jon Brock and colleagues have proposed “the temporal binding deficit hypothesis of autism”, suggesting that many features of autism may arise due to failures to temporally integrate information across a wide range of brain areas [28,29]. Thus, the MN system may only be one candidate system for exploring the relationship between ASD, temporal disruption and EMR.

As yet, I can present no direct evidence that temporal disruption by EMR does influence MN development or that through this, or some other route (i.e. the cerebellum [30]), such disruption plays a role in ASD. A logical first step would be to examine prevalence rates in low versus high EMR environments. This could involve comparisons of local geographical areas, home environments or more/less developed nations. Of course, the pace of the current technological growth, particularly satellite communications, might make it extremely hard to find environments with no, or at least very low, EMR exposure. For such demographic studies to be successful it would also be important to improve the availability of measurement and visualization techniques for recording EMR in the environment.

It will also be important to provide theoretical or empirical evidence that temporal modulation can and does disrupt the development of the MN system. As a first step this might be achieved via computational modelling. That is, to provide proof-in-principle that the ability of two networks to coordinate with each other can be impaired by the presence of temporal noise. Existing network models of autism may provide a useful starting point for this work [31,32]. Empirically, while there have been many EMR studies involving adults, a closer examination of long-term effects of subtle temporal modulations of the nervous system, particularly in complex EMR environments, should be undertaken. Finally, the use of fMRI to perform non-invasive longitudinal studies of infant monkeys reared in high versus low EMR environments could provide a direct test of the current hypotheses in relation to the development of the MN system.

While I have been able to provide no definitive evidence for a link between ASD and EMR, my goal here is simply to point out that such a connection could exist and should, given the potential consequences, be taken seriously. At least one other paper has previously made this same connection [33]. Here, I hope to have taken additional steps in identifying a possible mechanism through which EMR could exert an influence, namely temporal disruption, and illustrating how an already implicated brain network might be particularly susceptible to such disruption. My hope is that this paper might prompt a re-evaluation of the temporal impact of

EMR on the *developing nervous system*, possibly helping to rule-out one potential environmental cause for the rise in ASD.

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